

Equine Viral Arteritis: Reproductive risks and management strategies in horse populations

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ABSTRACT

Equine Viral Arteritis (EVA) is an infectious disease in horses caused by Equine Arteritis Virus (EAV), a member of the Arterivirus genus in the Arteriviridae family. This disease has a significant impact on animal health and the horse breeding industry because it can cause systemic viremia, vascular damage, subcutaneous edema, abortion in pregnant mares, and persistent infection in adult stallions. Post-pubertal stallions can become androgen-dependent carriers, continuously excreting the virus through semen without showing clinical symptoms, thus acting as the main reservoir and route of venereal transmission. Transmission of the virus also occurs through respiratory and transplacental contact, with the risk of transmission increasing in dense populations, high mobility, and suboptimal reproductive management practices. This review summarized the latest literature on the characteristics of the virus, epidemiology, pathogenesis, immune response, clinical manifestations, diagnostics, and EVA control strategies. Detection of the virus through RT-qPCR and serological screening is the primary method for identifying acute cases and carriers, while selective vaccination and strict biosecurity measures have proven effective in suppressing the spread of the virus. The immune response to EAV involves innate and adaptive mechanisms, including the activation of macrophages, T cells, and the production of neutralizing antibodies, although it is not always able to eliminate the virus in the reproductive tissues of males. Overall, EVA control requires a multidimensional approach that integrates vaccination, carrier monitoring, reproductive management, and biosecurity. This information is important to support prevention strategies, outbreak control, and animal health policies in the global equine industry.

Introduction

Equine Viral Arteritis (EVA) is an infectious disease in horses caused by the Equine Arteritis Virus (EAV), a member of the Arterivirus genus in the Arteriviridae family (Ata *et al.*, 2023a). This disease is of significant relevance to animal health and the global horse breeding industry because it affects reproductive health, horse performance, and international trade (Bonsi *et al.*, 2023). EVA was first identified in the early 1950s in the United States, when outbreaks of respiratory disease and abortion occurred in Standardbred horses (Van Maanen *et al.*, 2025). Since then, the disease has been reported in various countries, including Europe, Asia, South America, and Australia, although its prevalence and spread patterns vary between regions (Baker *et al.*, 2022). The global distribution of EVA indicates that the disease is cosmopolitan, particularly in regions with advanced horse breeding and racing industries (Khaled and Besbaci, 2024).

EAV has a primary tropism for vascular endothelial cells, macrophages, and mononuclear phagocytic system cells, causing systemic viremia, vascular damage, and an intense immune response (Fosse *et al.*, 2021). Infection in adult horses is often mild or subclinical, but in pregnant mares, it can cause abortion, while adult stallions can become persistent carriers that excrete the virus through semen without clinical symptoms (Yang and Yang, 2021). These carrier stallions are the primary epidemiological source, allowing the virus to persist in the population even though most horses experience clinically limited infection (Franco *et al.*, 2025). EAV transmission occurs via respiratory, venereal, and transplacental routes, with direct or indirect exposure playing a role in virus spread (Balasuriya, 2014). The venereal route via semen from carrier stallions is the primary mechanism contributing to the geographical distribution of the disease, particularly in intensive breeding facilities (Perrett *et al.*, 2021).

The immune response to EAV involves complex interactions between innate and adaptive mechanisms (Wang and Shao, 2023). Activation of

phagocytes, macrophages, and natural killer (NK) cells produces proinflammatory cytokines such as Interleukin-1 (IL-1), Interleukin-6 (IL-6), Tumour Necrosis Factor alpha (TNF- α), and type I interferon, which play a role in controlling viral replication and the onset of early clinical symptoms (Mishra *et al.*, 2025). Cluster of designation 8 positive (CD8⁺) and cluster of designation 4 positive (CD4⁺) T cells and virus-specific neutralizing antibodies eliminate viremia and prevent systemic spread (Hsu *et al.*, 2022). However, in the reproductive tissues of males, the virus is able to evade immune elimination through local androgen-dependent mechanisms, allowing carrier stallions to remain long-term reservoirs (Hou *et al.*, 2025).

From a health management perspective, EVA poses significant challenges for the horse industry (Chapman *et al.*, 2024). Infection can lead to abortion, edema, and decreased performance, resulting in economic losses due to reproductive failure, restrictions on international trade, and outbreak control in breeding facilities (Serbessa *et al.*, 2023). Control strategies include strict biosecurity, serological and virological screening of stallions, selective vaccination, and proper reproductive management (Branda *et al.*, 2025). Live attenuated vaccines have been widely used to provide long-term protection, while inactivated vaccines are safer for sensitive populations, including pregnant female (Arora and Lakshmi, 2021). This integrated approach is essential to reduce virus prevalence and prevent further circulation in equine populations (Branda *et al.*, 2025).

Despite advances in molecular diagnostics, serological surveillance, and vaccination, which have improved EVA control capabilities, there are still significant gaps in research (Kurniawan *et al.*, 2025; Lakshmanan and Liu, 2025). Understanding the mechanisms of viral persistence, developing safer and more effective vaccines, and establishing population-based epidemiological models are necessary to strengthen global control strategies (Mody *et al.*, 2024). This review aimed to provide a scientific summary of the characteristics of the virus, epidemiology, pathogenesis,

transmission, immune response, and EVA control strategies, with the goal of providing a comprehensive scientific basis for researchers, veterinarians, and stakeholders in the equine industry.

Etiology

EVA is caused by the EAV, an RNA virus belonging to the Arteriviridae family, order Nidovirales (Franco *et al.*, 2025). The genus in which EAV is found is Arterivirus, which includes other important viruses such as Porcine Reproductive and Respiratory Syndrome Virus (PRRSV), Simian Hemorrhagic Fever Virus (SHFV), and Lactate Dehydrogenase-Increasing Virus (LDV) (Balasuriya *et al.*, 2013). This group of viruses shares biological similarities in terms of persistent infection in hosts, replication in monocyte-macrophage cells, and a tendency to cause vasculitis and partial immunosuppression (Ata *et al.*, 2023b; Kaps *et al.*, 2023).

EAV is an enveloped virus with a particle size of approximately 50–65 nm and has a positive-strand single-stranded RNA (ssRNA) genome of approximately 12.7 kb (Guo *et al.*, 2025). Its genome structure consists of two main regions: (1) the ORF1a/ORF1b region encoding non-structural polyproteins involved in viral replication, and (2) the ORF2–ORF7 region encoding structural proteins such as nucleocapsid (N), membrane glycoproteins (GP2, GP3, GP4), envelope protein (E), matrix protein (M), and major glycoprotein GP5 (Shin *et al.*, 2022; Zhang *et al.*, 2022). GP5 and M are important components in the formation of the envelope complex and mediate virus-host cell interactions (Feng *et al.*, 2022). GP5 also acts as the primary target for neutralizing antibodies, so changes in this protein can affect the virus's ability to evade the immune response (Luo *et al.*, 2023a).

This virus exhibits tropism for vascular endothelium, macrophages, and reproductive tract epithelial cells, particularly in males (Fosse *et al.*, 2021). Replication in endothelium and immune cells causes vascular damage and the release of inflammatory mediators that trigger edema, conjunctivitis, and fever in infected hosts (Ielapi *et al.*, 2021). The ability of EAV to form persistent infections occurs mainly in unvaccinated males, associated with testosterone hormonal regulation, which allows the virus to replicate in the accessory genital epithelium and be excreted through semen (Petrillo *et al.*, 2022). Thus, carrier males play a significant epidemiological reservoir role in the spread of the disease, especially in horse reproductive systems with high breeding intensity (Li *et al.*, 2024).

Genetic variability in EAV is reported to be quite high, although it generally produces only two clinical phenotype categories: strains with high virulence and strains with low virulence (Yamaoka and Ebihara, 2021). These differences are related to nucleotide variations in the GP5 gene, which have implications for differences in immunity levels and vaccine efficacy (Luo *et al.*, 2023b). Phylogenetic studies indicate the existence of a global clade with specific geographic distribution, related to international trade in horses and frozen semen (Radovic *et al.*, 2024). The EAV virus is relatively unstable to heat and lipid solvents, but can survive at low temperatures, including during semen cryopreservation, mak-

ing laboratory surveillance key to preventing reproductive transmission (Sharafi *et al.*, 2022).

History

EVA was first identified in 1953 in Ohio, United States, during an outbreak of respiratory disease and abortion in Standardbred horses (Timoney and McCollum, 1993). The causative virus was subsequently isolated and named EAV (Bhat *et al.*, 2025). Prior to this discovery, similar cases may have occurred in various regions, but were not clearly documented due to limited diagnostic facilities and understanding of the etiology of viral diseases in horses at that time.

Subsequent EVA outbreaks were reported in various countries, demonstrating the global distribution and epidemiological complexity of this disease (Qiao *et al.*, 2023). In the 1980s, significant outbreaks occurred in horse populations in Europe, including in the United Kingdom, Germany, and the Netherlands, characterized by an increase in acute respiratory cases, abortions in pregnant mares, and high morbidity in young horses (Macleay *et al.*, 2022). These events highlighted the importance of virological surveillance and biosecurity in the international horse trade, especially with the increased movement of racehorses at that time (Rendle *et al.*, 2023).

One outbreak that had major consequences for international regulations occurred in 2006 in Kentucky, United States—one of the world's largest horse breeding centers (Knox *et al.*, 2023). This outbreak was linked to the use of semen from EAV-carrier stallions that had not been previously diagnosed, resulting in widespread transmission through artificial insemination (Mahmoud *et al.*, 2023). This incident highlighted the important role of carrier stallions in the epidemiology of the disease and prompted the implementation of systematic virological testing of stallions in many countries as a requirement in breeding programs (Branda *et al.*, 2025).

In addition to the United States and Europe, EVA outbreaks have also been reported sporadically in Asia, South America, and Australia (Brouwer *et al.*, 2021; Qiao *et al.*, 2023). However, there are variations in the intensity of outbreaks between countries, influenced by reproductive management practices, quarantine systems, vaccination rates, and local diagnostic capacity. In some countries, EVA remains a notifiable disease, reflecting its potential economic impact, particularly on the world-class horse breeding industry.

Epidemiology

EVA has a wide global distribution and has been detected in equine populations in various parts of the world, although the prevalence rate and pattern of spread vary between regions (Knox and Beddoe, 2021). The disease is most commonly reported in North America and Europe, particularly in countries with advanced horse breeding and racing industries (Stallones *et al.*, 2023). In the United States, EVA has long been

Table 1. Global distribution and epidemiology of EVA.

Region	Prevalence / Affected Population	Related Species / Breeds	Control/Supervision Strategy	Important Notes	References
North America	High; routine clinical and subclinical cases	Standardbred, Thoroughbred, Quarter Horse	Breeding stock screening, semen use regulation, intensive monitoring	Sporadic cases continue to occur during the mating season.	(Junior <i>et al.</i> , 2021)
Europe	Relatively high serological prevalence	Warmblood, Standardbred	Testing of stallions before breeding, control of horse and semen imports	Variations in policy between countries; the mobility of competition horses facilitates cross-border movement	(Cavalleri <i>et al.</i> , 2022)
Asia	Data has increased over the past two decades	The racing population in Japan and China	Supervision of large breeding facilities	Developing countries (Southeast Asia) lack surveillance; likely underdiagnosed	(Steinbach <i>et al.</i> , 2015)
South America	Limited; EAV antibodies detected in several countries	Local horses and industrial breeding	Limited; systematic surveillance is rare	Limited clinical case data	(Desanti-Consoli <i>et al.</i> , 2022)
Africa	Very limited; confirmed cases in South Africa	Local horse	Minimal; limited diagnostic facilities	Many countries do not report; actual distribution may not have been identified	(Resende <i>et al.</i> , 2022)

recognized as an important health threat to horses, especially Standardbred, Thoroughbred, and Quarter Horse breeds, with clinical and sub-clinical infections reported routinely (Ata *et al.*, 2023a). Intensive monitoring programs, screening of stallions, and strict regulations on semen use contribute to outbreak control, although sporadic cases still occur, especially during the breeding season when reproductive interactions increase (Khan *et al.*, 2025). Table 1 presents a summary of the geographical distribution of EVA based on prevalence, affected horse populations, frequently infected breeds, control or surveillance strategies implemented, and important epidemiological notes.

In Europe, this virus is also a widespread pathogen, with relatively high serological prevalence in Warmblood and Standardbred horses (Cavalleri *et al.*, 2022). Countries such as Germany, France, Italy, the Netherlands, and the United Kingdom have implemented comprehensive surveillance policies that include testing stallions before they are used in breeding programs and strict regulations on the importation of horses and frozen semen (Pacchiarotti *et al.*, 2022). However, variations in policy implementation between countries and the high mobility of horses in international competition still provide opportunities for cross-border spread of the virus, especially in unvaccinated populations (Timoney, 2007).

In Asia, EVA surveillance data show an increase in reporting over the past two decades. Japan and China have reported virus circulation and implemented surveillance measures at large breeding facilities, especially those connected to the racing industry (Branda *et al.*, 2025). In contrast, developing countries in Southeast Asia, including Indonesia, still show limited epidemiological information (Steinbach *et al.*, 2015). The lack of scientific publications and serological surveillance raises the suspicion that EVA may be an underdiagnosed disease, especially in small farms or breeding facilities without standard biosecurity procedures (Freddi *et al.*, 2025).

Meanwhile, information on EVA in South America and Africa is still relatively limited (Junior *et al.*, 2021; Desanti-Consoli *et al.*, 2022). Brazil and Argentina have detected EAV antibodies in horse populations, but clinical case reports and systematic surveillance data are still rare (Bezerra *et al.*, 2022). In Africa, infections have been confirmed in several equine populations in South Africa, but most countries on the continent have not reported the presence of the virus, most likely due to limited diagnostic facilities and low surveillance priorities for equine reproductive diseases (Resende *et al.*, 2022). This situation indicates that the actual distribution of EVA in the region may not yet be fully identified (Qiao *et al.*, 2023).

Pathogenesis

The pathogenesis of EVA is a complex process that reflects the interaction between Equine Arteritis Virus EAV and the host immune system and vascular tissue (Mahmoud *et al.*, 2023). This virus has a primary tropism for vascular endothelial cells, macrophages, and cells of the mononuclear phagocytic system, so the course of the disease is dominated by systemic vascular disorders, inflammation, and changes in the reproductive system, especially in adult males (Zarate-Sanchez *et al.*, 2024).

Infection begins with the entry of the virus through the respiratory mucosa or venereal route (Jagirdhar *et al.*, 2023). In respiratory transmission, the virus binds to specific receptors on the epithelial cells of the upper respiratory tract and alveolar macrophages, followed by local replication (Clementi *et al.*, 2021). In venereal transmission, the virus reaches the reproductive tract and then infects the epithelial cells of the male accessory glands (Teixeira *et al.*, 2021). Within a short time, the virus spreads to regional lymphoid tissue, particularly the tonsils and mandibular lymph nodes, where primary replication occurs and triggers viremia (Brisse and Hickman, 2025).

The viremia phase is a critical stage in pathogenesis, allowing the virus to spread systemically through the bloodstream (Xu *et al.*, 2024). EAV tropism for endothelial cells causes diffuse vascular damage through direct cytolysis and endothelial dysfunction due to inflammatory responses

(Hennigs *et al.*, 2021). Pathological changes include increased vascular permeability, interstitial edema, and inflammatory infiltration, which underlie clinical signs such as edema of the extremities, scrotum, and periorbital area, as well as effusion into body cavities (Wang *et al.*, 2024).

In addition to vascular damage, EAV triggers activation of innate and adaptive immune responses, including the release of proinflammatory cytokines such as IL-1, IL-6, and TNF- α , which contribute to fever and systemic malaise (Rabaan *et al.*, 2021). In some animals, an excessive immune response can exacerbate tissue damage through immunopathological mechanisms (Aichele *et al.*, 2022). In pregnant horses, placental infection and vascular damage to the uterus and fetus can cause abortion, especially in the middle to late trimesters of pregnancy (Li *et al.*, 2024). Abortion in EVA is associated with fetal hypoxia and intrauterine vascular lesions, although direct damage to fetal tissue may also occur (Zhao *et al.*, 2021).

In adult males, pathogenesis follows a unique pathway characterized by persistent infection of the accessory reproductive glands, especially the seminal vesicles (Dutta *et al.*, 2024). The precise regulation of the virus in these tissues is androgen-dependent, occurring only in post-pubertal males with adequate testosterone levels (Abdelhamid and Mazor, 2025). The virus maintains long-term persistence through local immune evasion mechanisms, without causing significant tissue damage, so that carrier males remain healthy but become sources of virus through semen (Teixeira *et al.*, 2021).

The formation of neutralizing antibodies and an effective T-lymphocyte response usually accompanies recovery from acute infection (Mancuso *et al.*, 2022). However, although long-term immunity can develop, EAV can avoid total elimination in some stallions through local adaptation in the reproductive system (Amat *et al.*, 2021). This makes carrier stallions a key component in EVA epidemiology and a primary target for control strategies (Ata *et al.*, 2023b). The pathogenesis of Equine Viral Arteritis is illustrated in Figure 1, showing the sequential progression from viral entry through systemic viremia to vascular damage and clinical outcomes.

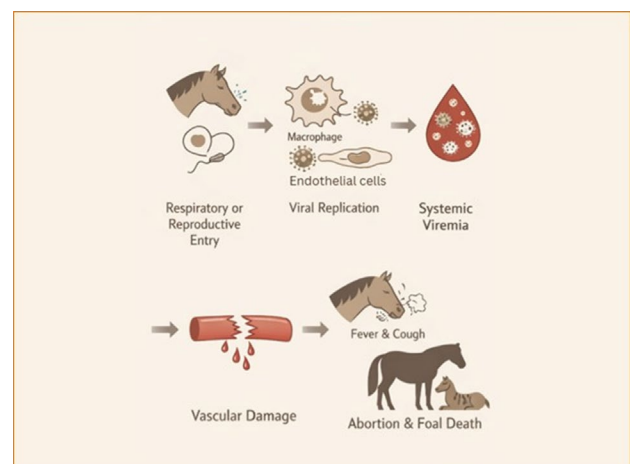


Figure 1. Pathogenesis of EVA.

Pathology

The pathology of EVA reflects the nature of the virus as an arterivirus with a primary tropism for vascular endothelial cells and immune system tissues (Balasuriya *et al.*, 2013). The pathological changes that occur are mainly related to systemic vascular damage, edema, and widespread inflammation, which ultimately contribute to clinical manifestations such as fever, subcutaneous edema, conjunctivitis, and abortion in pregnant mares (Del Piero, 2000). The pathological picture can be seen at both the macroscopic and microscopic levels, depending on the phase of infection and the organs involved (Chen and Pan, 2021).

Macroscopically, the most commonly observed lesions are edema and congestion in the subcutaneous tissue, particularly in the extremi-

ties, scrotum, and periorbital region (Timoney and McCollum, 1993). Serous effusions are often found in the pleural, peritoneal, and pericardial cavities due to increased vascular permeability and endothelial damage (Karpathiou *et al.*, 2022). Lymphadenopathy and splenomegaly may occur as a response to systemic immune stimulation. In severe cases, petechial hemorrhages and ecchymoses may be seen in mucosal and serosal tissues, reflecting severe systemic vasculitis (López and Martinson, 2017).

In animals that experience abortion, the placenta often shows severe edema, congestion, and areas of focal necrosis (Fedorka *et al.*, 2022). Aborted fetuses usually appear edematous with visceral congestion, especially in the lungs and liver (Ulum *et al.*, 2021). However, fetal lesions can vary from minimal to severe, depending on the gestational age at the time of infection and the maternal immune response (Megli and Coyne, 2022).

Histopathologically, proliferative and necrotizing vasculitides are characteristic features of EVA pathology (Ishizu *et al.*, 2023). Vascular lesions include degeneration and necrosis of endothelial cells, mononuclear and neutrophilic inflammatory cell infiltration of the vessel walls, and proliferation of smooth muscle cells and fibroblasts (Sheikh *et al.*, 2024). Endothelial damage leads to increased vascular permeability, edema formation, and fibrin deposition in the vessel lumen, which can cause local thrombosis (Wautier and Wautier, 2022). In addition to vasculitis, lymphocyte and macrophage infiltration are found in various organs, including the lungs, liver, kidneys, and spleen (Leone *et al.*, 2021).

In the lungs, common lesions include interstitial pneumonia with alveolar edema, congestion, and inflammatory cell infiltration (Carvalho and Stevenson, 2022). In some cases, there are areas of atelectasis or interstitial emphysema due to gas exchange dysfunction (Morris *et al.*, 2025). In the liver, changes may range from mild to moderate hepatocyte degeneration with portal inflammatory infiltration (Dhingra *et al.*, 2022). In the reproductive tract of male carriers, pathological changes are minimal, but the virus persists in the accessory gland epithelium, particularly the seminal vesicles, reflecting ongoing infection without significant tissue damage (Guiton and Drevet, 2023).

Clinical manifestations

The clinical manifestations of EVA vary widely, ranging from subclinical infection to pronounced systemic symptoms, depending on the virulence of the virus strain, the animal's immune status, age, and physiological condition, especially in stallions and pregnant mares (Mahmoud *et al.*, 2023). Most infected adult horses exhibit mild to moderate symptoms, and in some cases, the infection may be asymptomatic, especially in animals that have acquired immunity from previous infections or vaccination (Glaser *et al.*, 1997). However, in susceptible individuals such as young horses, stressed horses, and pregnant mares, the infection can cause more severe clinical disease and potentially lead to significant reproductive losses (Li *et al.*, 2024).

The early clinical phase is generally characterized by acute high fever, often reaching 41°C, accompanied by systemic signs such as lethargy, anorexia, and decreased performance (Holyoak *et al.*, 2008). Respiratory manifestations are one of the main features, including conjunctivitis, excessive lacrimation, serous to mucopurulent rhinitis, and cough (Balasuriya, 2014). Edema is one of the pathognomonic symptoms that reflects vascular damage due to the tropism of the virus to the endothelium (Odendaal *et al.*, 2021). Edema usually occurs in the extremities, preputium, scrotum, and ventral abdomen, and may be accompanied by urticaria and small hemorrhagic lesions on the mucous membranes (Timoney and McCollum, 1993). More severe cases may present with dyspnea, ataxia, and regional lymph node swelling as a systemic inflammatory response (Auyezkhankyzy *et al.*, 2024).

In males, EVA can affect the reproductive system by causing orchitis, epididymitis, and scrotal swelling, which in some cases leads to temporary semen quality decline and decreased libido (Bao *et al.*, 2025). Al-

though most males can recover from fertility disorders, some individuals can become persistent carriers and continue to excrete the virus through semen without showing further clinical symptoms (Guo *et al.*, 2024). This situation has important epidemiological implications, especially in breeding stallion populations, as carrier males are the main source of disease transmission through the venereal route (Górecka-Bruzda *et al.*, 2023).

In pregnant mares, infection can cause abortion, especially in the late trimester of pregnancy (Leon *et al.*, 2023). Abortion often occurs suddenly without preceding severe maternal symptoms, and the expelled fetus generally does not show characteristic macroscopic lesions, so the diagnosis can be missed without laboratory testing (Agerholm *et al.*, 2021). In addition to abortion, subclinical infection in mares can also interfere with pregnancy success through uterine inflammation that disrupts implantation and early embryonic development (Pascottini *et al.*, 2023).

In foals and young horses, EVA tends to produce a more severe form of the disease, especially if they do not yet have protective maternal antibodies (Eertink *et al.*, 2024). The infection can progress to interstitial pneumonia and enteritis, which in certain cases can be fatal due to dehydration and respiratory failure (Pozzessere *et al.*, 2024). Mortality in this group, although rare in populations with good health management, remains a concern on breeding farms with large juvenile populations (Stojanovic *et al.*, 2022).

Diagnosis

Diagnosis of EVA requires an integrated approach that combines clinical evaluation, reproductive history, and laboratory confirmation, given that the clinical symptoms of this disease are nonspecific and may resemble other respiratory or reproductive infections in horses (Kaps *et al.*, 2024). Clinically, the presence of fever, conjunctivitis, mild epistaxis, peripheral edema, and respiratory distress may raise initial suspicion, but a definitive diagnosis can only be obtained through detection of the causative agent or specific immune response to EAV (Ata *et al.*, 2023b). A history of exposure to carrier stallions, the use of uncertified frozen semen, or the occurrence of abortions within a horse population are epidemiological factors that support the indication for laboratory testing (Ruiz-Saenz, 2010).

Laboratory diagnostic methods for EVA consist of serological testing, nucleic acid detection, virus isolation, and cell culture testing (Srivastava and Prasad, 2023). Serological testing is the most widely used approach for monitoring infection, particularly through the virus neutralization test (VNT), which is considered the gold standard for detecting neutralizing antibodies against EAV (Knox and Beddoe, 2021). In addition, the enzyme-linked immunosorbent assay (ELISA) technique has been widely adopted due to its rapid and practical nature, although interpretation of results requires caution, especially in vaccinated horses, as the vaccination antibody response may not distinguish between natural infection and immunization. Therefore, repeat testing at intervals of two to three weeks to observe antibody titer increases is often recommended in questionable cases (Kölling and Langemeyer, 2023).

For direct detection of pathogens, real-time reverse transcription polymerase chain reaction (RT-qPCR) is the most sensitive and specific method, enabling identification of viral RNA from nasopharyngeal, blood, fetal tissue, and semen samples (Dikaman *et al.*, 2025). This test is crucial for detecting carrier males, who often show no clinical symptoms but continue to shed the virus through semen over the long term (Guo *et al.*, 2024). Virus isolation in horse kidney cell culture remains the confirmatory method in reference laboratories, although its use is increasingly being replaced by RT-qPCR due to the longer time required and high laboratory requirements (Dikman *et al.*, 2025).

In cases of abortion, examination of fetal and placental tissue is important, with histopathological evaluation often showing vasculitis and inflammatory lesions, although these findings are not specific to EVA (Beça *et al.*, 2024). Therefore, combining histopathological examination

with molecular techniques is the optimal diagnostic strategy (Kurniawan *et al.*, 2025). In general, the diagnostic approach to EVA should take into account vaccination status, exposure history, and stage of infection, so that the interpretation of results is more accurate and can support reproductive health management decisions (Teixeira *et al.*, 2021).

Transmission

EVA caused by EAV has a complex transmission pattern and is influenced by horse population dynamics, reproductive systems, and environmental factors (Parrish *et al.*, 2021). The virus can spread through two main routes, namely horizontal respiratory transmission and venereal transmission, with male carrier horses playing an important role as a source of long-term infection (Balasuriya *et al.*, 2013).

Respiratory transmission is the primary mechanism during the acute outbreak phase. Transmission occurs through inhalation of droplets or aerosols containing the virus, which are excreted by infected horses during the viremic phase (Lv *et al.*, 2021). Respiratory secretions such as nasal discharge, saliva, and respiratory aerosols are the dominant sources of transmission, especially in crowded environments such as breeding stables, training facilities, and racetracks (Ruiz-Saenz, 2010). Direct contact between horses or exposure through contaminated fomites, including stable and transportation equipment, also contributes to the spread of the virus, although the virus is relatively unstable in extreme environmental conditions and is susceptible to disinfectants (Dayaram *et al.*, 2021).

In addition, venereal transmission plays a central role in the persistence of the virus in equine populations (Vissani *et al.*, 2021). The virus can be transmitted through natural insemination or artificial insemination using fresh, chilled, or frozen semen from carrier stallions (De Clercq *et al.*, 2021). EAV can persistently establish itself in post-pubertal stallions through a mechanism dependent on androgen hormones, particularly testosterone, making these individuals biological reservoirs that can continuously excrete the virus through semen without showing clinical signs (Al-Kass and Morrell, 2024). Mares infected through the reproductive tract can develop viremia and subsequently transmit the virus through the respiratory route to other horses in the same environment (Balasuriya *et al.*, 2013).

Although less common, transplacental transmission can also occur, resulting in abortion in pregnant mares (Leon *et al.*, 2023). The virus that penetrates the placenta causes fetal death due to systemic vascular damage and fetal hypoxia, making abortion one of the important clinical manifestations of EAV infection (Yu *et al.*, 2021). Additionally, transmission through indirect contact with other bodily fluids or contaminated reproductive instruments can also occur, although its contribution is relatively small compared to the primary routes (Leung, 2021).

Environmental variables, population density, and horse mobility play a role in accelerating transmission (Otzdorff *et al.*, 2021). International trade in horses and semen distribution without adequate diagnostic controls has been linked to several major outbreaks in various countries (Bonsi *et al.*, 2023). Therefore, controlling EAV transmission requires a multidisciplinary approach, including routine serological testing, identification of carrier stallions, reproductive biosecurity practices, and targeted vaccination of high-risk horse populations (Branda *et al.*, 2025). The transmission pathways of Equine Arteritis Virus (EAV) in horse populations are illustrated in Figure 2, highlighting respiratory transmission between horses, venereal transmission via infected semen, and transplacental transmission from pregnant mares to the fetus.

Risk factors

EVA has an epidemiological dynamic that is influenced by various risk factors related to the host, environment, and horse management system (Franco *et al.*, 2025). Understanding these factors is very important in dis-

ease prevention, early detection, and control efforts, given their potential impact on animal health, reproductive performance, and international trade in horses and semen (Zuidema *et al.*, 2021).

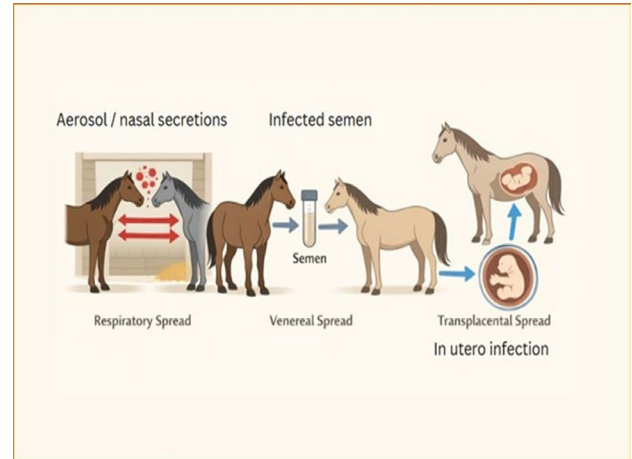


Figure 2. Transmission routes of EAV in horses.

One of the main risk factors is the presence of carrier stallions that are persistently infected with EAV (Otzdorff *et al.*, 2021). This persistent infection is androgen-dependent, so it is more common in adult stallions than in young horses or mares (Ata *et al.*, 2023a). Carrier stallions continuously excrete the virus through semen without showing clinical symptoms, making them a key epidemiological reservoir in the horse population (Otzdorff *et al.*, 2021). The use of semen from carrier stallions in artificial insemination or natural breeding programs significantly increases the potential for virus spread, especially in large-scale breeding operations (Zuidema *et al.*, 2021).

Horse population density and intensive husbandry practices also play an important role in accelerating virus transmissibility through respiratory contact (Laing *et al.*, 2021). Horses placed in dense environments, such as training centers, racing facilities, or collective stables, are more susceptible to exposure to respiratory droplets containing the virus (Duba *et al.*, 2024). The movement or transport of horses between regions, especially in competition or international trade, increases the risk of introducing the virus to new areas when biosecurity procedures are not optimally implemented (Grewar *et al.*, 2021).

Another significant risk factor is inadequate vaccination programs and serological surveillance (Carcelen *et al.*, 2025). Unvaccinated horse populations or those that have never been exposed to the virus are more susceptible to outbreaks, especially if carrier individuals are present in the population (Oladunni *et al.*, 2021). In addition, the lack of semen testing and serological status in stallions can result in undetected spread of the virus through reproductive routes (Mahmoud *et al.*, 2023).

Reproductive status and pregnancy also need to be considered, as pregnant mares are at risk of abortion due to EAV infection, especially if exposed during the critical gestation phase (Ruby and Janes, 2023). Meanwhile, environmental factors, such as poor sanitation and inadequate air circulation, although not the primary source of the virus, can increase aerosol stability and facilitate respiratory exposure in susceptible populations (Ijaz *et al.*, 2023).

Economic impact

EVA has a significant economic impact on the equine industry, particularly in the breeding sector, international trade, and horse competitions (Mahmoud *et al.*, 2023). Economic losses are mainly related to reproductive disorders, decreased performance, restrictions on animal movement, and disease control and surveillance costs (Kappes *et al.*, 2023).

Abortion in pregnant mares is one of the most economically damaging clinical consequences, as it can lead to the loss of high-quality offspring in breeding programs (Bechert *et al.*, 2022). This has a direct im-

pact on the decline in breeding farm productivity and the loss of potential income from the sale of high-value foals (Arora and Lakshmi, 2021). In addition, infection in adult stallions can result in persistent carrier status, which has serious implications for reproductive management (Otzdorff et al., 2021). Carrier stallions often require intensive testing programs, restrictions on semen use, or even removal from breeding programs, thereby increasing maintenance costs and reducing the economic value of the animal (Khan et al., 2025).

Indirect losses also arise due to reduced performance of infected racehorses or competitions, even though clinical symptoms in most horses are subclinical (MacLachlan and Balasuriya, 2006). Decreased stamina, fever, and edema can affect training and competition schedules, leading to potential loss of race prizes and sponsors (Timoney and McCollum, 1993). In addition, EVA outbreaks often result in domestic and international trade restrictions on live horses, frozen semen, and embryos, especially for countries or regions that have not implemented routine vaccination programs and EVA-free certification. These restrictions slow the flow of equine commodities, hampering the economic growth of related sectors (Singh et al., 2021).

Additional costs also arise from the implementation of biosecurity, vaccination, and diagnostic programs, including RT-qPCR and serological testing for stallion screening (Kurniawan et al., 2025). While these measures are essential to prevent the spread of the virus, their long-term costs can burden small and medium-sized breeding facilities (Dikman et al., 2025). Overall, EVA imposes a substantial economic burden, primarily through reproductive disruption, performance decline, and trade barriers, making sustained surveillance, selective vaccination, and strict reproductive management essential in minimizing losses in the global horse industry (Cruz-Lopez et al., 2017).

Vaccination

The EVA vaccination program is a strategic component in disease control efforts, especially in horse populations with high genetic and economic value (Mahmoud et al., 2023). Vaccination aims to minimize the risk of clinical infection, prevent abortion in pregnant horses, and inhibit virus circulation in the population by reducing the number of susceptible individuals (Li et al., 2024). Two types of vaccines commonly used internationally are live attenuated vaccines and inactivated vaccines, with the choice of vaccine type tailored to epidemiological conditions, breeding objectives, and local animal health policies (Glaser et al., 1997).

Live attenuated vaccines have been shown to provide a strong immune response and long-term protection, especially in stallions and non-pregnant mares (Ahmed et al., 2023). However, this vaccine is not recommended for use in pregnant mares and very young foals due to the potential risk to the fetus and the immature immune system (Desanti-Consoli et al., 2022). In contrast, inactivated vaccines are safer for use in sensitive groups such as pregnant mares, although the immune response generated tends to be more moderate and requires a stricter booster schedule to maintain protective immunity (Arora and Lakshmi, 2021).

An effective vaccination program requires initial serological testing, especially in male horses intended for breeding (Kaps et al., 2024). Vaccinated stallions must have a seronegative record prior to the first vaccination to distinguish antibodies resulting from vaccination from antibodies resulting from natural infection (Allkofer et al., 2021). Documentation of serological status is very important in the international trade of semen and live animals to avoid regulatory barriers and ensure that there is no confusion in the identification of carrier stallions (Egyptien et al., 2023).

Young horses are generally given primary vaccination at around 6–12 months of age, adjusted for maternal antibody status, which can affect the immune response to the vaccine (Allkofer et al., 2021). Booster vaccines are given according to the manufacturer's protocol or veterinary authority guidelines, usually every 6–12 months, depending on the risk of exposure (Barbosa et al., 2022). In mares intended for breeding, vacci-

nation is recommended prior to pregnancy to ensure optimal protection and prevent abortions due to acute infection (Li et al., 2024).

In addition to individual protection, vaccination plays an important epidemiological role in creating herd immunity, especially in areas with high horse mobility or intensive breeding facilities (Rykala et al., 2025). However, vaccination does not replace biosecurity protocols such as semen testing, isolation of new horses, and monitoring of carrier stallions (Thompson et al., 2022). The integration of vaccination, strict reproductive management, and continuous serological surveillance is an effective foundation for EVA control, helping to break the cycle of venereal and respiratory transmission while reducing the potential for outbreaks (Timoney and McCollum, 1993).

Control

Control of EVA requires an integrated approach that includes biosecurity strategies, reproductive management, serological surveillance, and selective vaccination (Kaps et al., 2024). Control efforts are designed to prevent the introduction of the virus into virus-free populations, suppress spread in endemic populations, and minimize the reproductive and economic impacts caused by EAV infection (Balasuriya et al., 2013). The complexity of EVA control is mainly influenced by the presence of carrier stallions, which can persistently excrete the virus through semen without showing clinical symptoms, thereby potentially triggering secondary venereal and respiratory transmission (Ata et al., 2023b).

Biosecurity is a key component in EVA control. This practice includes isolating new horses for 21–28 days, restricting direct contact between individuals from different epidemiological groups, and implementing strict sanitation procedures in facilities and equipment (Thompson et al., 2022). In addition, monitoring horse movements, including the implementation of health certification and serological status requirements prior to transportation or participation in competitions, is an important step in preventing cross-regional spread (Knox and Beddoe, 2021).

Strict reproductive management is essential to prevent transmission through semen (Kaya et al., 2021). Stallions must be tested serologically and virologically before entering the breeding program, and semen from carrier stallions should only be used in closed systems with clear risk documentation and vaccination of recipient females to prevent clinical manifestations (Đuračka et al., 2023). Artificial insemination protocols using controlled semen offer a higher chance of preventing transmission compared to natural mating, but still require verification of virus-free status (De Clercq et al., 2021).

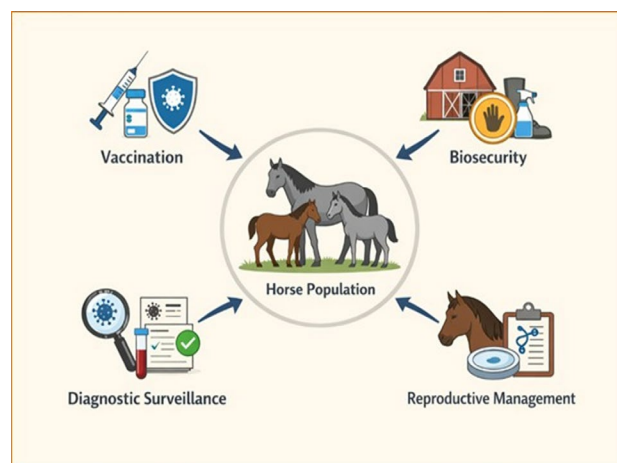


Figure 3. Integrated Control Strategies for EVA.

Additionally, epidemiological surveillance and case reporting play a role in early detection and rapid response to outbreaks (Meckawy et al., 2022). Laboratory testing, including serology and molecular detection, enables the identification of acute cases and carriers, allowing control measures such as isolation, movement restrictions, and contact tracing to

be implemented in a timely manner (Chung *et al.*, 2021). Educating horse owners, veterinarians, and reproduction facility managers is also a key pillar in improving compliance with animal health protocols (MacLachlan and Balasuriya, 2006). An integrated control framework for Equine Viral Arteritis, encompassing vaccination, biosecurity, diagnostic surveillance, and reproductive management, is illustrated in Figure 3.

Conclusion

EVA is a viral disease with global distribution that affects equine reproductive health and the breeding industry. The virus causes systemic viremia, vascular damage, and abortion in pregnant mares, while adult stallions can become persistent androgen-dependent carriers. Transmission occurs via respiratory, venereal, and transplacental routes, with carrier stallions serving as the primary reservoir. Innate and adaptive immune responses play a role in controlling infection, but do not always eliminate the virus in carrier stallions. Effective control strategies include strict biosecurity, serological and virological screening of stallions, selective vaccination, and proper reproductive management.

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Conflict of interest

The authors declare that there is no conflict of interest.

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